Overview

Product name: Human Factor H ELISA Kit
Detection method: Colorimetric

Precision

<table>
<thead>
<tr>
<th>Sample</th>
<th>n</th>
<th>Mean</th>
<th>SD</th>
<th>CV%</th>
</tr>
</thead>
<tbody>
<tr>
<td>Overall</td>
<td></td>
<td></td>
<td></td>
<td>4.8%</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Sample</th>
<th>n</th>
<th>Mean</th>
<th>SD</th>
<th>CV%</th>
</tr>
</thead>
<tbody>
<tr>
<td>Overall</td>
<td></td>
<td></td>
<td></td>
<td>9.9%</td>
</tr>
</tbody>
</table>

Sample type: Cell culture supernatant, Saliva, Milk, Urine, Serum, Plasma, Cerebral Spinal Fluid
Assay type: Sandwich (quantitative)
Sensitivity: > 0.25 ng/ml
Range: 81% - 114%
Recovery: 97%
Assay time: 4h 00m
Assay duration: Multiple steps standard assay
Species reactivity: Reacts with: Human

Product overview

Abcam’s Factor H Human in vitro ELISA (Enzyme-Linked Immunosorbent Assay) kit is designed for the quantitative measurement of Human Factor H in urine, saliva, milk, plasma, serum, cerebrospinal fluid and cell culture supernatants.

A Factor H specific antibody has been precoated onto 96-well plates and blocked. Standards or test samples are added to the wells and subsequently a Factor H specific biotinylated detection antibody is added and then followed by washing with wash buffer. Streptavidin-Peroxidase Conjugate is added and unbound conjugates are washed away with wash buffer. TMB is then used to visualize Streptavidin-Peroxidase enzymatic reaction. TMB is catalyzed by Streptavidin-Peroxidase to produce a blue color product that changes into yellow after adding acidic stop solution. The density of yellow coloration is directly proportional to the amount of Factor H.
The entire kit may be stored at -20°C for long term storage before reconstitution - Avoid repeated freeze-thaw cycles.

**Platform**

Microplate

**Properties**

**Storage instructions**

Store at -20°C. Please refer to protocols.

**Components**

<table>
<thead>
<tr>
<th>Component</th>
<th>Count</th>
</tr>
</thead>
<tbody>
<tr>
<td>100X Streptavidin-Peroxidase Conjugate</td>
<td>1 x 80µl</td>
</tr>
<tr>
<td>10X Diluent M Concentrate</td>
<td>1 x 30ml</td>
</tr>
<tr>
<td>20X Wash Buffer Concentrate</td>
<td>2 x 30ml</td>
</tr>
<tr>
<td>70X Biotinylated Human Factor H Antibody</td>
<td>1 x 90µl</td>
</tr>
<tr>
<td>Chromogen Substrate</td>
<td>1 x 8ml</td>
</tr>
<tr>
<td>Factor H Microplate (12 x 8 well strips)</td>
<td>1 unit</td>
</tr>
<tr>
<td>Factor H Standard</td>
<td>1 vial</td>
</tr>
<tr>
<td>Sealing Tapes</td>
<td>3 units</td>
</tr>
<tr>
<td>Stop Solution</td>
<td>1 x 12ml</td>
</tr>
</tbody>
</table>

**Function**

Factor H functions as a cofactor in the inactivation of C3b by factor I and also increases the rate of dissociation of the C3bBb complex (C3 convertase) and the (C3b)NBB complex (C5 convertase) in the alternative complement pathway.

**Tissue specificity**

Expressed by the liver and secreted in plasma.

**Involvement in disease**

Genetic variations in CFH are associated with basal laminar drusen (BLD) [MIM:126700]; also known as drusen of Bruch membrane or cuticular drusen or grouped early adult-onset drusen.

Drusen are extracellular deposits that accumulate below the retinal pigment epithelium on Bruch membrane. Basal laminar drusen refers to an early adult-onset drusen phenotype that shows a pattern of uniform small, slightly raised yellow subretinal nodules randomly scattered in the macula. In later stages, these drusen often become more numerous, with clustered groups of drusen scattered throughout the retina. In time these small basal laminar drusen may expand and ultimately lead to a serous pigment epithelial detachment of the macula that may result in vision loss.

Defects in CFH are the cause of complement factor H deficiency (CFHD) [MIM:609814]. A disorder that can manifest as several different phenotypes, including asymptomatic, recurrent bacterial infections, and renal failure. Laboratory features usually include decreased serum levels of factor H, complement component C3, and a decrease in other terminal complement components, indicating activation of the alternative complement pathway. It is associated with a number of renal diseases with variable clinical presentation and progression, including membranoproliferative glomerulonephritis and atypical hemolytic uremic syndrome.
Defects in CFH are a cause of susceptibility to hemolytic uremic syndrome atypical type 1 (AHUS1) [MIM:235400]. An atypical form of hemolytic uremic syndrome. It is a complex genetic disease characterized by microangiopathic hemolytic anemia, thrombocytopenia, renal failure and absence of episodes of enterocolitis and diarrhea. In contrast to typical hemolytic uremic syndrome, atypical forms have a poorer prognosis, with higher death rates and frequent progression to end-stage renal disease. Note=Susceptibility to the development of atypical hemolytic uremic syndrome can be conferred by mutations in various components of or regulatory factors in the complement cascade system. Other genes may play a role in modifying the phenotype.

Genetic variation in CFH is associated with age-related macular degeneration type 4 (ARMD4) [MIM:610698]. ARMD is a multifactorial eye disease and the most common cause of irreversible vision loss in the developed world. In most patients, the disease is manifest as ophthalmoscopically visible yellowish accumulations of protein and lipid (known as drusen) that lie beneath the retinal pigment epithelium and within an elastin-containing structure known as Bruch membrane.

Sequence similarities
Contains 20 Sushi (CCP/SCR) domains.

Cellular localization
Secreted.

Images

Standard curve with background signal subtracted (duplicates; +/- SD).

Factor H measured in culture supernatants (tested at dilution range 1/1-1/30; duplicates +/- SD).
Factor H measured in biological fluids (duplicates +/- SD). Human serum and plasma were tested at dilution range of 1/50000-1/1500000. Other biological fluids were tested at 1/1-1/500 and gave measurable values (milk: 400, saliva: 200, CSF: 2000 and urine: 40 ng per mL).

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